

FOOD INTAKE OF RATS WITH PREOPTIC LESIONS

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Brobeck suggested that in homeothermic animals the body temperature is one of the factors which regulates the food intake (1). Experimental evidences supporting his view are many, such as his own observation that when environmental temperature rises, the food intake decreases in many species of animals (2); Stevenson's observation that food intake preserves a normal deep body temperature of activity-restricted rats (3); Young's observation that starvation causes a lower rectal temperature in dogs (4); and the well known effect of the specific dynamic action of foodstuff.

Since adult men and animals maintain their body weight at constant level for years in spite of great variations in energy expenditure, their food intake must have been adjusted to their heat loss. There is good reason to speculate therefore, that the regulations of both food intake and body temperature must be closely related somehow.

One way of testing the relationship of food intake to body temperature regulations is to produce lesions in the ventromedial nucleus of the hypothalamus to induce increased intake of food (hyperphagia), to see whether there is any impairment of temperature regulation. Conversely, lesions may be produced in the preoptic area of the hypothalamus to cause impairment of heat loss mechanism (hyperthermia), to see whether there is any impairment of regulation of food intake.

Previous experiments revealed that the temperature regulating mechanism is not impaired in hyperphagic rats (5). This paper reports the findings on the food intake of rats with preoptic lesions.

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MATERIALS AND METHODS

Twenty eight male Sprague-Dawley rats weighing 250-300 g were housed in individual cages with food and water *ad lib*. Non-spilling food cups were used to insure accurate measurement of food consumption.

Colonic temperatures were measured daily by inserting a rectal thermometer at least 4 cm from the anus. Readings were made 2 minutes after the insertion. Colonic temperatures were always measured first when rats were attended every morning.

Room temperature was maintained for 4 days at 70 F, and then for 4 days at 80 F and for 4 days at 90 F and 94 F subsequently. When these four 4-day measurements were obtained, the room temperature was then maintained at 80 F, which is considered to be the neutral environmental temperature and was the usual temperature for the laboratory unless otherwise specified.

Using the stereotaxic instrument, bilateral electrolytic lesions were placed in the preoptic area of the hypothalamus (A 9.0, L 0.5, H 1.0) with 2 ma d-c for 15 seconds. Rats receiving sham operations which included the same surgical procedures without the insertion of electrodes served as controls.

Two weeks after the operation, food intake and colonic temperatures were again measured at different environmental temperatures following the same preoperative schedule.

RESULTS

Preoperative Results

The food intake of rats at different environmental temperatures is shown in Table I. It shows the well known inhibitive effect of raised environmental temperature on food intake. 28 animals were used for this experiment.

TABLE I
*Influence of environmental temperature
on food intake*

Room temp., F	Food intake (g/day/rat)
70	23.7±3.1*
80	21.0±2.3
90	17.6±2.5
94	12.5±2.9

* mean±s.d.

Postoperative Results

After lesions were made, more than half of the rats died within 24 hours. Acute pulmonary edema was observed in most of the cases. Those who survived and the control rats were studied two weeks after the operation. The results are shown in Table II.

The food intake of lesioned rats was not significantly different from that of the control rats over the environmental temperature range of 70-90 F. A high environmental temperature of 94 F proves fatal to all lesioned rats.

The colonic temperature of lesioned rats was significantly higher than that of the controls under all three temperature conditions.

DISCUSSION

In this study, over half of the rats with bilateral lesions in the preoptic area between the optic chiasma and the anterior commissure developed acute pulmonary edema and died. This finding corroborates the original observation of Maire and Patton (6).

Rats with preoptic lesions ate the same

amount of food as the control animals did, but the colonic temperature of the lesioned rats was significantly higher. This indicates that the deep body temperature does not directly affect the feeding nor the satiety centers of the hypothalamus. In both lesioned and control rats, their food intake decreased when the environmental temperature increased from 70 to 90 F. It is interesting to note that their deep body temperature remained the same, although their food intake was decreased. This fact again supports the view that it is not the deep body temperature *per se* which affects the hypothalamic feeding or satiety centers to account for decreased food intake during heat stress.

Lesions in the preoptic area caused impairment of heat loss mechanism in rats. Nevertheless, such rats were shown to retain some degree of their temperature regulating mechanism (7). The fact that they were able to maintain constant body temperature although at a higher level, in face of graded elevations of temperature from 70 to 90 F, supports this concept.

When rats were exposed to a temperature of 94 F, all lesioned ones were found dead when they were attended the next morning. Although their colonic temperatures were not measured before death, it is believed that the cause of death was hyperthermia as reported before (7). Hamilton also found that this temperature is the critical temperature for rats bearing such lesions (8).

Colonic temperatures of control rats also elevated significantly when the environmental temperature was raised from 90 to 94 F. This fact indicates that an environmental temperature of 94 F is a strong heat stress in which rats can no longer maintain their normal deep body temperature.

TABLE II
Food intake and colonic temperature in rats with preoptic lesions

Room temp., F	Food intake (g/day/rat)		Colonic temp., F	
	Lesioned (9)†	Control (7)	Lesioned (9)	Control (7)
70	24.5±2.8	25.0±3.0	101.4±0.8*	100.2±0.5
80	21.7±2.5	20.5±2.4	101.4±0.8*	99.9±0.6
90	17.4±2.7	16.8±2.9	101.5±0.9*	99.8±0.6
94	Rats died	13.0±3.3	Rats died	101.4±0.8**

† Number of rats.

* P<0.05 when compared with controls.

** P<0.05 when compared with body temperature of controls at other environmental temperatures.

This experiment indicates that the regulation of food intake is not affected in rats whose heat loss mechanism is impaired. Previously reported experiment (5) showed that the regulation of body temperature is not impaired when rats were made hyperphagic. The available data therefore suggest that the normal functioning of one center does not require the existence of the other center, or to put it another way, that the destruction of one center does not necessarily affect the function of the other to any noticeable extent.

SUMMARY

The food intake of rats with preoptic lesions was studied in cool (70 F), neutral (80 F) and warm (90 F) environments. The food intake of lesioned rats was comparable to that of the normal controls despite their higher deep body temperature. The relationship between food intake and body temperature was discussed.

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